WEST Search History

DATE: Wednesday, July 10, 2002

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=OR		
L7	L6 and (dr3 or dr4)	8	L7
L6	L5 and (class adj II)	48	L6
L5	mhc and (gad or (glucose adj dehydrogenase))	81	L5
L4	L3 and (gad or (glucose adj dehydrogenase))	4	L4
L3	L2 and mhc	66	L3
L2	(stahl)[IN] OR (schendel)[IN] or (meinl)[in] or (endl)[in] or (albert)[in] or (jung)[in] or (dornmair)[in]	67421	L2
L1	(stahl)[IN] OR (schendel)[IN]	3345	L1

END OF SEARCH HISTORY

Print Request Result(s)

Printer Name: cm1_9e12_gblbptr
Printer Location: cm1__9e12

- US005945401: Ok
- US005830682: Ok
- US006218132: Ok
- US005648219: Ok
- US005624895: Ok
- US006060309: Ok
- US005824315: Ok

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                  frequency
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 NEWS  6  Mar 08  Gene Names now available in BIOSIS
 NEWS  7  Mar 22  TOXLIT no longer available
 NEWS  8  Mar 22  TRCTHERMO no longer available
 NEWS  9  Mar 28  US Provisional Priorities searched with P in CA/CAplus
                  and USPATFULL
 NEWS 10  Mar 28  LIPINSKI/CALC added for property searching in REGISTRY
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 NEWS 17  Apr 22  BIOSIS Gene Names now available in TOXCENTER
 NEWS 18  Apr 22  Federal Research in Progress (FEDRIP) now available
 NEWS 19  Jun 03  New e-mail delivery for search results now available
 NEWS 20  Jun 10  MEDLINE Reload
 NEWS 21  Jun 10  PCTFULL has been reloaded
 NEWS 22  Jul 02  FOREGE no longer contains STANDARDS file segment

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               CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
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=> file medline caplus embase biosis
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

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=> s endl J?/au or schendel D?/au or meinl E?/au
L1          709 ENDL J?/AU OR SCHENDEL D?/AU OR MEINL E?/AU

=> s l1 or stahl P?/au or albert W?/au or jung G?/au or dornmair K?/au
L2         6339 L1 OR STAHL P?/AU OR ALBERT W?/AU OR JUNG G?/AU OR DORNMAIR
                K?/AU

=> s mhc
L3       113257 MHC

=> s l2 and mhc
L4          400 L2 AND MHC

=> s l4 and (GAD or dehydrogenase)
L5            2 L4 AND (GAD OR DEHYDROGENASE)

=>

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6            2 DUP REM L5 (0 DUPLICATES REMOVED)

=> dis l6 1-2 ibib abs

L6   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         1997:155018  CAPLUS
DOCUMENT NUMBER:          126:156406
TITLE:                    Peptides and peptide derivatives from glutamic acid
                          decarboxylase for the early diagnosis and treatment of
                          type I diabetes
```

INVENTOR(S): **Endl, Josef**; **Stahl, Peter**;
Albert, Winfried; **Schendel, Dolores**;
Boitard, Christian; van Endert, Peter; **Jung, Guenther-Gerhard**
PATENT ASSIGNEE(S): Boehringer Mannheim Gmbh, Germany
SOURCE: Ger. Offen., 16 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 19525784	A1	19970116	DE 1995-19525784	19950714
WO 9704085	A1	19970206	WO 1996-EP3093	19960715
W: JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 839191	A1	19980506	EP 1996-925751	19960715
R: AT, CH, DE, ES, FR, GB, IT, LI				
JP 10511985	T2	19981117	JP 1996-506274	19960715

PRIORITY APPLN. INFO.: DE 1995-19525784 19950714
WO 1996-EP3093 19960715

AB Peptides and their derivs. obtained from glutamic acid decarboxylase (**GAD**) are described, which are used alone or in complexes with class II **MHC** mols. for the detection of a predisposition to diabetes, and for the treatment of diabetes by building up an immune tolerance to **GAD**. Thus, **GAD**-specific T cells were established from peripheral blood lymphocytes from type I diabetics, cultured, and their proliferative response to recombinant human **GAD** and **GAD**-derived peptides was studied.

L6 ANSWER 2 OF 2 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1997:439533 BIOSIS
DOCUMENT NUMBER: PREV199799738736
TITLE: High affinity presentation of an autoantigenic peptide in type I diabetes by an HLA class II protein encoded in a haplotype protecting from disease.
AUTHOR(S): Bach, Jean-Marie; Otto, Heike; Nepom, Gerald T.; **Jung, Guenther**; Cohen, Helene; Timsit, Jose; Boitard, Christian; Van Endert, Peter M. (1)
CORPORATE SOURCE: (1) INSERM U25, 161 Rue Sevres, 75743 Paris Cedex 15 France
SOURCE: Journal of Autoimmunity, (1997) Vol. 10, No. 4, pp. 375-386.
ISSN: 0896-8411.
DOCUMENT TYPE: Article
LANGUAGE: English

AB Polymorphism of the genes coding for the human histocompatibility leukocyte antigen class II DR and DQ molecules makes the single largest genetic contribution to the risk of developing insulin-dependent diabetes mellitus (IDDM) and can be associated with highly elevated as well as decreased disease frequency. The mechanism of IDDM risk modification by HLA polymorphism is likely to involve differential presentation of autoantigenic peptides by HLA class 11 proteins. We have generated T cell lines (TCL) with specificity for the IDDM autoantigen 65 kDa glutamic acid decarboxylase (GAD65) from lymphocytes of two patients carrying HLA class 11 alleles associated with distinct risk of IDDM (DRB1*0101/0401 and 1302/1501). For both patients, TCL generated at various time points all recognized single epitopes mapped as **GAD** 88-99 and 248-257, respectively. These epitopes are presented by the DRB1*0101 and DRB5*0101, HLA class 11 molecules associated with a moderately elevated risk of IDDM, or carried in a strongly protective haplotype, respectively. In an HLA/peptide binding assay, epitope **GAD** 248-257 was shown to possess high affinity for DRB5*0101. This epitope overlaps with a central **GAD** pep tide binding to the high risk allele DQBL *0302 and containing a Coxsackie P2C-identical mimicry sequence, raising the possibility of competition of DRB5*0101 and DQB1*0302 for binding of a central GAD65 fragment.

```
=> s mhc
L7      113257 MHC

=> s MHC (P) (class (1N) II)
L8       47183 MHC (P) (CLASS (1N) II)

=> s DR3 or DR4
L9       15792 DR3 OR DR4

=> s l8 (P) l9
L10        946 L8 (P) L9

=> s l10 (P) (GAD or (glucose (1N) dehydrogenase))
L11         12 L10 (P) (GAD OR (GLUCOSE (1N) DEHYDROGENASE))

=> dup rem l11
PROCESSING COMPLETED FOR L11
L12           4 DUP REM L11 (8 DUPLICATES REMOVED)

=>
Connection closed by remote host

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  NEWS 17  Apr 22  BIOSIS Gene Names now available in TOXCENTER
  NEWS 18  Apr 22  Federal Research in Progress (FEDRIP) now available
  NEWS 19  Jun 03  New e-mail delivery for search results now available
  NEWS 20  Jun 10  MEDLINE Reload
  NEWS 21  Jun 10  PCTFULL has been reloaded
  NEWS 22  Jul 02  FOREGE no longer contains STANDARDS file segment

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                AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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=>
=> file medline caplus embase biosis
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     5.67       5.67

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=> s MHC (P) (class (1N) II)
L1       47192 MHC (P) (CLASS (1N) II)

=> s l1 (P) (GAD or (glucose (1N) dehydrogenase))
L2          74 L1 (P) (GAD OR (GLUCOSE (1N) DEHYDROGENASE))

=> s l2 and dr4
L3           9 L2 AND DR4

=> s l2 and (drb1 0401)
L4           0 L2 AND (DRB1 0401)

=> s l2 and (drbi?
UNMATCHED LEFT PARENTHESIS 'AND (DRBI?'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s l2 and (drbi?)
L5           0 L2 AND (DRBI?)

=> dup rem l3
PROCESSING COMPLETED FOR L3
L6           3 DUP REM L3 (6 DUPLICATES REMOVED)

=> dis l6 1-3 ibib abs

L6   ANSWER 1 OF 3  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER:   2001:267766  BIOSIS
DOCUMENT NUMBER:    PREV200100267766
TITLE:              Role of DM in regulating the presentation of a diabetes
                    autoantigen.
AUTHOR(S):          Jayne, Jennifer A. (1); Lich, John D. (1); Blum, Janice S.
                    (1)
CORPORATE SOURCE:   (1) Microbiology and Immunology, Indiana University School
                    of Medicine, 635 Barnhill Drive, MS255, Indianapolis, IN,
                    46202 USA
SOURCE:             FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A675.
                    print.
                    Meeting Info.: Annual Meeting of the Federation of American
                    Societies for Experimental Biology on Experimental Biology
                    2001 Orlando, Florida, USA March 31-April 04, 2001
                    ISSN: 0892-6638.
DOCUMENT TYPE:      Conference
LANGUAGE:           English
SUMMARY LANGUAGE:   English
AB   Glutamate decarboxylase (GAD) is a key autoantigen targeted
     during the development of insulin-dependent diabetes mellitus (IDDM).
     Presentation of GAD epitopes in the context of HLA-DR and DQ
     alleles may be important in disease initiation as well as the induction of
     tolerance to this self-protein. MHC-restricted presentation of
     GAD has been reported to vary among APC from different
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individuals, suggesting a genetic factor may regulate the display of **GAD** peptides in the context of **class II** proteins for T cell recognition. Studies have also indicated that B lymphocytes play an important role in antigen presentation during the development of IDDM. To examine the mechanisms modulating **GAD** presentation, the presentation of **GAD** epitopes in the context of HLA-**DR4** was examined in a panel of human B-lymphoblastoid cell lines. Differential **class** II-restricted presentation of **GAD** epitopes was observed using these cell lines. This difference was not linked to the expression of other DR alleles, but rather to another set of **MHC**-encoded proteins. These **MHC** -encoded proteins differentially regulated both exogenous and endogenous **GAD** presentation. Studies are underway to further define the importance of these proteins in modulating **GAD** epitope presentation.

L6 ANSWER 2 OF 3 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000253238 MEDLINE
DOCUMENT NUMBER: 20253238 PubMed ID: 10790426
TITLE: Cytoplasmic processing is a prerequisite for presentation of an endogenous antigen by major histocompatibility complex class II proteins.
AUTHOR: Lich J D; Elliott J F; Blum J S
CORPORATE SOURCE: Department of Microbiology and Immunology and the Walther Oncology Center, Indiana University School of Medicine, Indianapolis, Indiana 46202, USA.
CONTRACT NUMBER: T32DK07519 (NIDDK)
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (2000 May 1) 191 (9) 1513-24.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200005
ENTRY DATE: Entered STN: 20000613
Last Updated on STN: 20000613
Entered Medline: 20000530

AB Biochemical and functional studies have demonstrated major histocompatibility complex (**MHC**) **class II** -restricted presentation of select epitopes derived from cytoplasmic antigens, with few insights into the processing reactions necessary for this alternate pathway. Efficient presentation of an immunodominant epitope derived from glutamate decarboxylase (**GAD**) was observed regardless of whether this antigen was delivered exogenously or via a cytoplasmic route into human histocompatibility leukocyte antigen **class II-DR4**(+) antigen-presenting cells. Presentation of exogenous as well as cytoplasmic **GAD** required the intersection of **GAD** peptides and newly synthesized **class II** proteins. By contrast, proteolytic processing of this antigen was highly dependent upon the route of antigen delivery. Exogenous **GAD** followed the classical pathway for antigen processing, with an absolute requirement for endosomal/lysosomal acidification as well as cysteine and aspartyl proteases resident within these organelles. Presentation of endogenous **GAD** was dependent upon the action of cytoplasmic proteases, including the proteasome and calpain. Thus, translocation of processed antigen from the cytoplasm into membrane organelles is necessary for **class II** -restricted presentation via this alternate pathway. Further trimming of these peptides after translocation was mediated by acidic proteases within endosomes/lysosomes, possibly after or before **class II** antigen binding. These studies suggest that processing of exogenous and cytoplasmic proteins occurs through divergent but overlapping pathways. Furthermore, two cytoplasmic proteases, the proteasome and calpain, appear to play important roles in **MHC class II** -restricted antigen presentation.

L6 ANSWER 3 OF 3 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999333721 MEDLINE
DOCUMENT NUMBER: 99333721 PubMed ID: 10403912
TITLE: T cell response pattern to glutamic acid decarboxylase 65 (GAD65) peptides of newly diagnosed type 1 diabetic patients sharing susceptible HLA haplotypes.
AUTHOR: Rharbaoui F; Mayer A; Granier C; Bouanani M; Thivolet C; Pau B; Orgiazzi J; Madec A M
CORPORATE SOURCE: CNRS-UMR9921, Faculte de Pharmacie, Montpellier, France.
SOURCE: CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (1999 Jul) 117 (1) 30-7.
Journal code: 0057202. ISSN: 0009-9104.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990806
Last Updated on STN: 19990806
Entered Medline: 19990728

AB Autoantibodies and autoreactive T lymphocytes directed against several pancreatic beta cell proteins such as GAD65 have been identified in the circulation before and at the onset of clinical type 1 (insulin-dependent) diabetes. Using GAD65 synthetic peptides, we studied the proliferative response of peripheral blood mononuclear cells (PBMC) either from recently diagnosed type 1 diabetic patients, of whom the majority share the disease-associated HLA **class II** haplotype (**DR4** -DQB1*0201 or DR3-DQB1*0302), or from HLA-matched control subjects. We found that 67% (14/21) of the type 1 diabetic patients and 39% (9/23) of the control subjects exhibited a positive proliferative response. Compared with control subjects, however, PBMC from diabetic patients proliferated more frequently ($P < 0.05$) in the presence of peptide pools from the C-terminal region of GAD65 (amino acids 379-585). Diabetic patients with the same HLA-DQ or HLA-DR alleles showed partially identical T cell reactivity, but no clear correlation could be made between **MHC class II** specificity and T cell epitopes because of multiple combinations of **class II** alleles. In addition, by flow cytometry, we studied the direct binding of GAD65 peptides to **MHC class II** molecules of Epstein-Barr virus (EBV)-transformed B (EBV-B) cells obtained from a diabetic patient. We found that 11 **GAD** peptides were able to bind to the highly susceptible haplotype DRB1*0301/0401-DQA1*0301/0501-DQB1*0302/0201 on the surface of EBV-B cells in partial correlation with the results obtained in the proliferation assays.

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=> dis his

     (FILE 'HOME' ENTERED AT 17:27:17 ON 10 JUL 2002)

     FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 17:43:19 ON 10 JUL 2002
L1         47192 S MHC (P) (CLASS (1N) II)
L2            74 S L1 (P) (GAD OR (GLUCOSE (1N) DEHYDROGENASE))
L3             9 S L2 AND DR4
L4             0 S L2 AND (DRB1 0401)
L5             0 S L2 AND (DRBI?)
L6             3 DUP REM L3 (6 DUPLICATES REMOVED)

=> end
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:y
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                26.43      32.10

STN INTERNATIONAL LOGOFF AT 17:50:22 ON 10 JUL 2002
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9

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FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 17:43:19 ON 10 JUL 2002

L1 47192 S MHC (P) (CLASS (1N) II)
L2 74 S L1 (P) (GAD OR (GLUCOSE (1N) DEHYDROGENASE))
L3 9 S L2 AND DR4
L4 0 S L2 AND (DRB1 0401)
L5 0 S L2 AND (DRBI?)
L6 3 DUP REM L3 (6 DUPLICATES REMOVED)

Kaufman
Native 366, 69-71

4-5 ⇒ pool

4-5 8

SQ 2

DR4

DR1 5

1 1

101 = [illegible] 47,51,52 110F 48,49-54

1/2 lst w/ DR5

112nd index w/ndeclaration

ancestor

b

plant